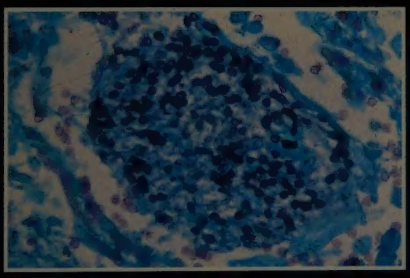
Color Atlas of Respiratory Disease



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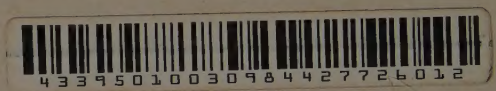

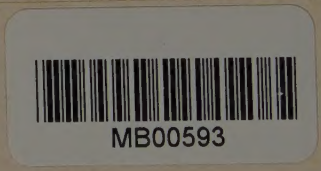

Color Atlas of Respiratory Disease

Volume 4 of 4

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GP Greenwich Press
Greenwich Connecticut

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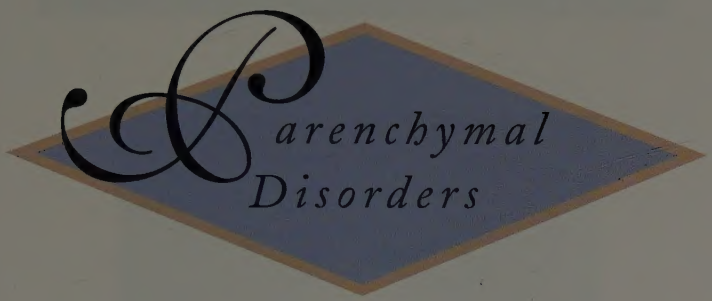
Introduction

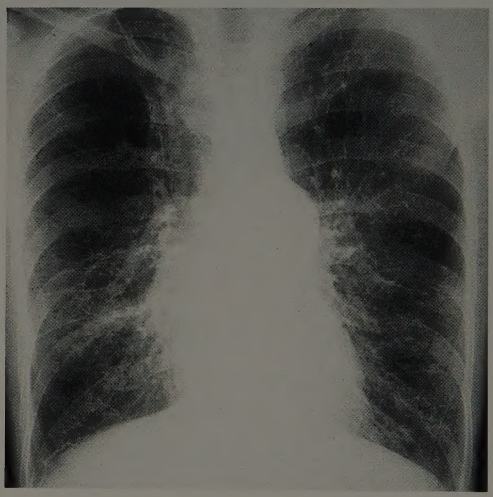
Respiratory disorders represent a diverse group of diseases that pose a diagnostic challenge for the clinician. *The Color Atlas of Respiratory Disease,* a four-volume atlas, includes images of direct or indirect visualization of the respiratory tract. Radiographs, computed tomography and magnetic resonance imaging, and bronchoscopy views, as well as cytology and biopsy slides and ECG tracings, are presented to assist the clinician in making a diagnosis.

A wide variety of pulmonary disorders are included within the four volumes: Volume 1, diseases of the upper respiratory tract and pulmonary vascular disease; Volume 2, airway diseases; Volume 3, respiratory tract infections; and Volume 4, parenchymal and pleural disorders. Different presentations of the disorder and/or its progression are shown. The captions describe the image and its interpretation rather than the disease and its etiology and management.

Volume 4 focuses on parenchymal disorders such as chronic interstitial, occupational, and collagen vascular diseases, and pleural space disorders. It is hoped that this atlas will prove valuable to you in your practice of clinical medicine.


Parenchymal
Disorders

Chronic Interstitial Pulmonary Disorders



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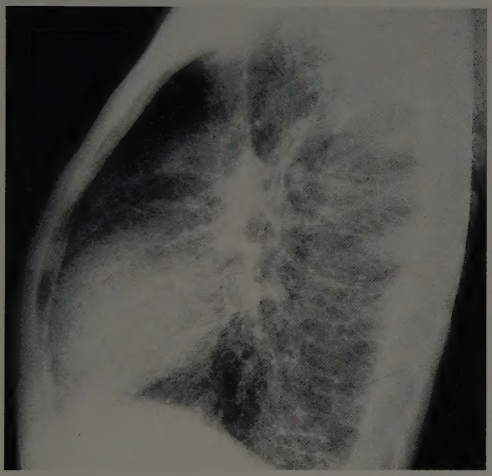

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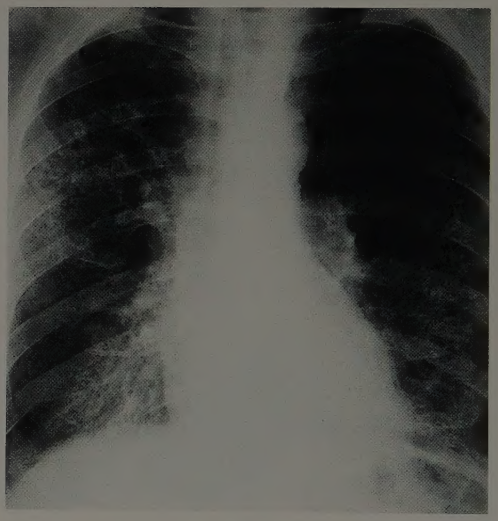
1–2 ♦ Desquamative interstitial pneumonitis. Posterior-anterior and lateral views show linear irregular opacities predominantly at the bases and midlung fields.



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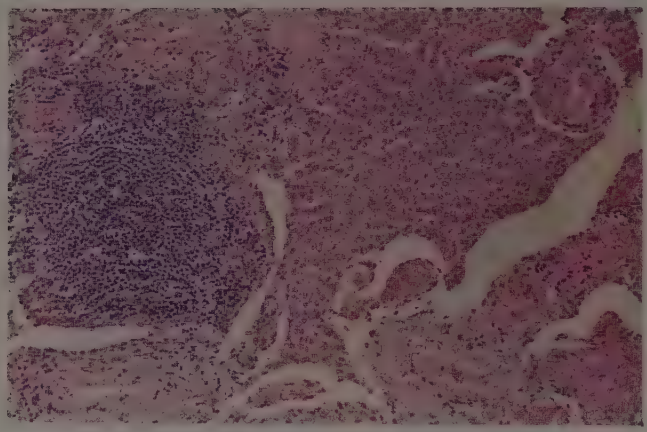
3 ♦ Desquamative interstitial pneumonitis before treatment.

4

4 ♦ Desquamative interstitial pneumonitis after a dramatic response to steroid therapy.



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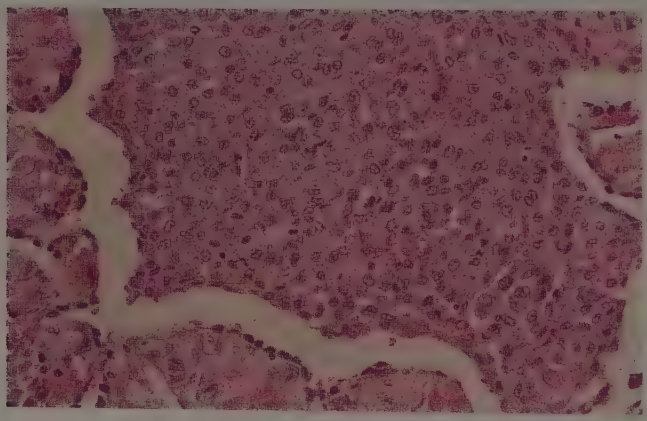

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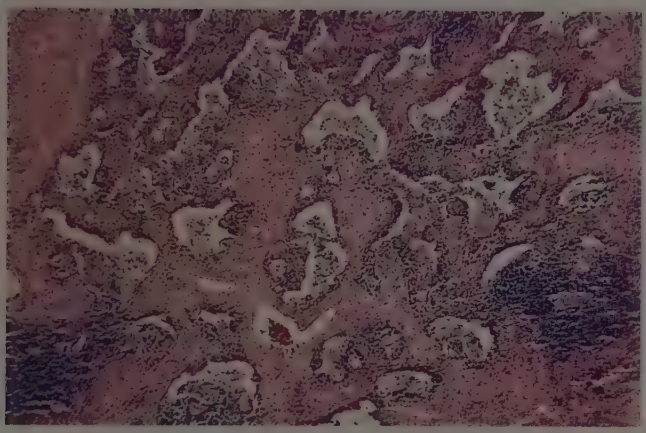
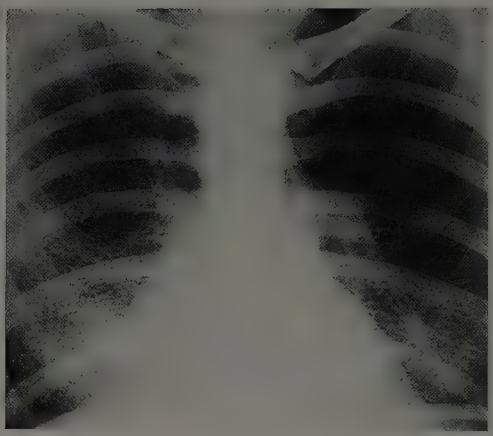


7

5–6 ♦ Interstitial pulmonary fibrosis is a condition of chronic inflammation of the alveolar area with subsequent destruction and progressive fibrosis. Pathologically, there is thickened alveolar septa, chronic alveolitis, dilitation of the alveolar ducts, and increased smooth muscle in the interstitium. In 5 and 6 an earlier phase of desquamative interstitial pneumonitis is seen.

7 ♦ A later phase of desquamative interstitial pneumonitis.



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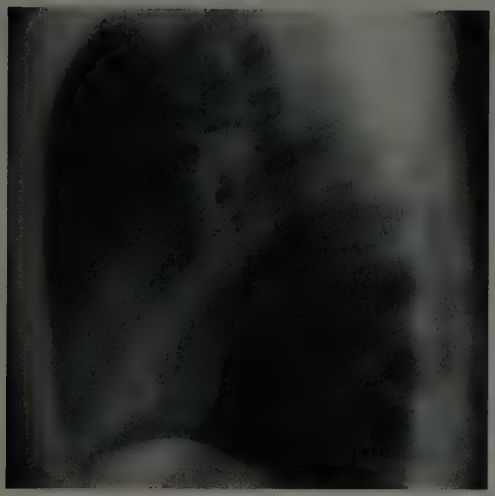
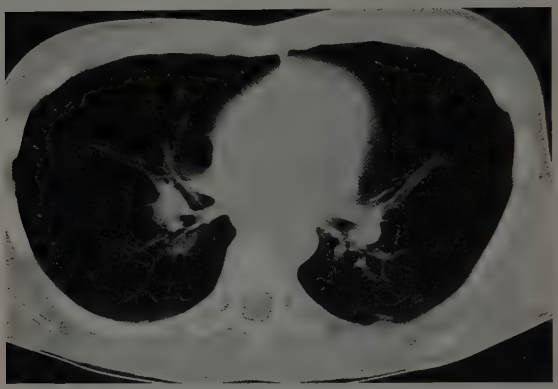


10

8 ♦ Bilateral, symmetric, diffuse, fine reticulo-reticulonodular patterns of eosinophilic granuloma. "Honeycomb" lung can also be seen on chest radiographs. Increased lung volumes are seen; in most fibrotic diseases decreased lung volumes are noted.

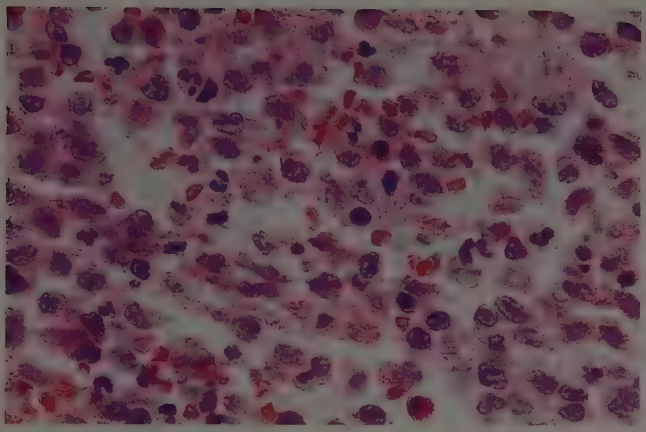
9–10 ♦ Hyperinflation and a fine reticulo-reticulonodular pattern are demonstrated in the posterior-anterior and lateral views. The patient is a 17-year-old smoker who presented at two different times with spontaneous pneumothoraces due to eosinophilic granuloma.



11

11 ♦ The finer detail of the chest computed tomography demonstrates multiple blebs. This classic presentation of eosinophilic granuloma is due to rupture of subpleural blebs leading to pneumothoraces.

12–13 ♦ Primary pulmonary histiocytosis X is one of a group of diseases that includes eosinophilic granuloma. The hallmark of the disease is the proliferation of Langerhans cells known as histiocytosis X cells.



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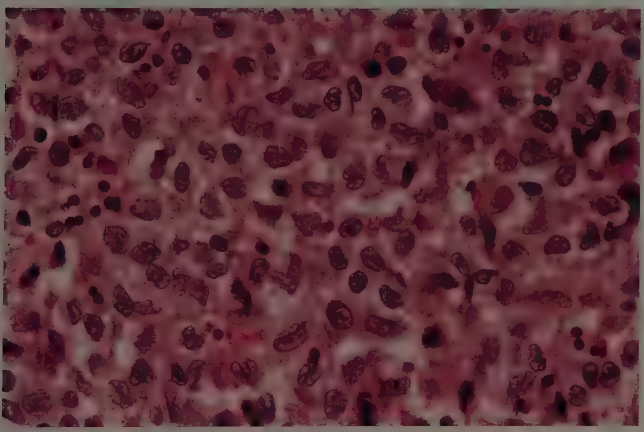

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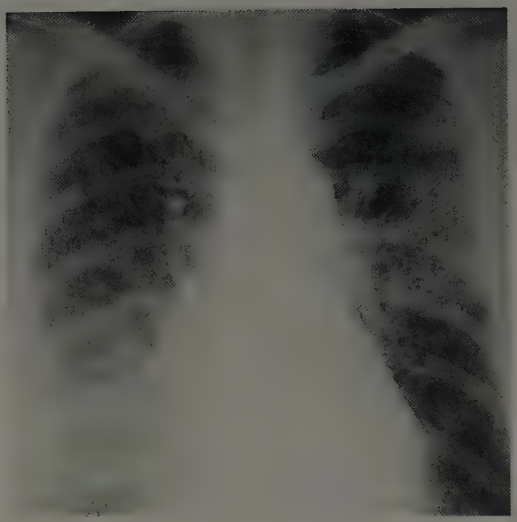
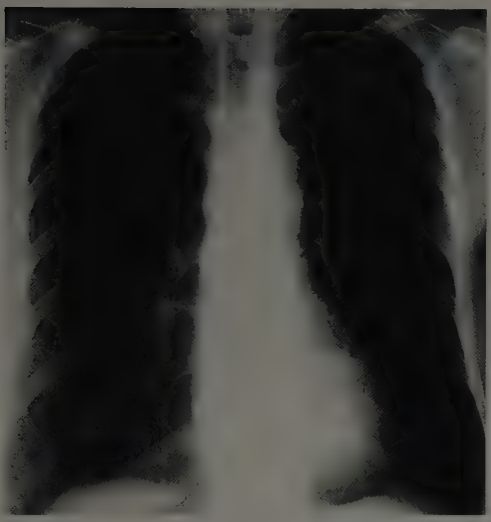


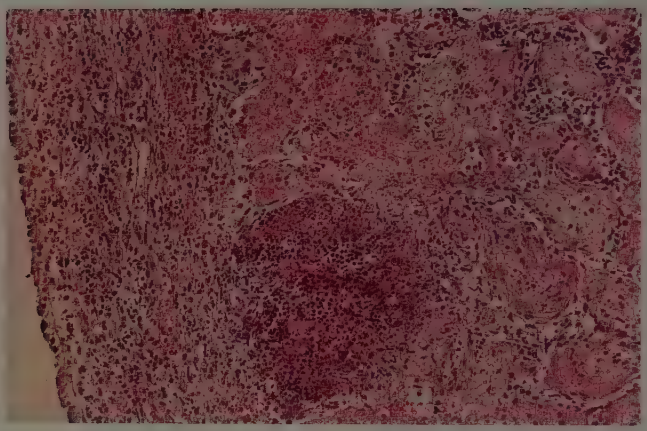
14

14 ♦ Chronic eosinophilic pneumonia. Bilateral homologous alveolar lung infiltrates are shown. The characteristic radiographic findings are peripheral, patchy, alveolar infiltrates, with ill-defined margins and relative sparing of the central portions of the lungs. This distinctive pattern is known as "photographic negative of pulmonary edema."



15

15 ♦ A dramatic response to steroid therapy administered for chronic eosinophilic pneumonia.



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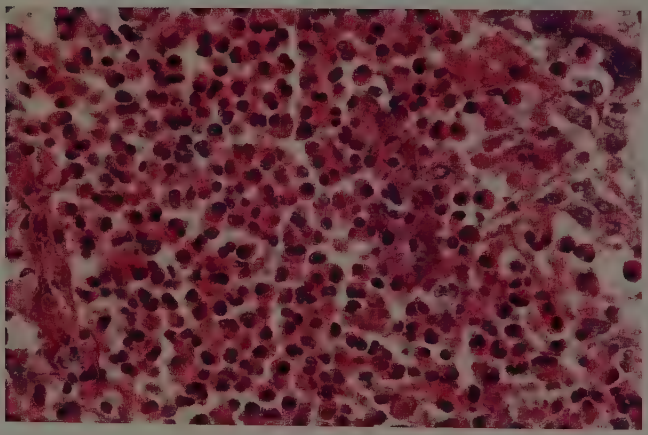

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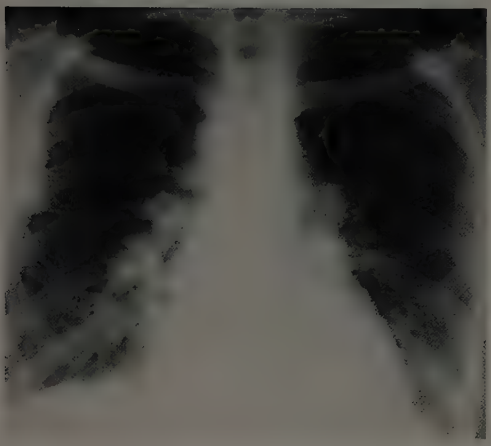
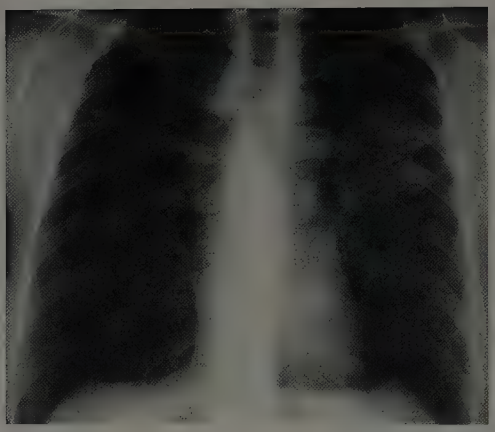


18

16–17 ♦ Alveolar exudate consisting of eosinophils and mononuclear cells consistent with eosinophilic pneumonia.

18 ♦ Sarcoidosis. Hilar node enlargement and a reticulonodular pattern of lung disease.



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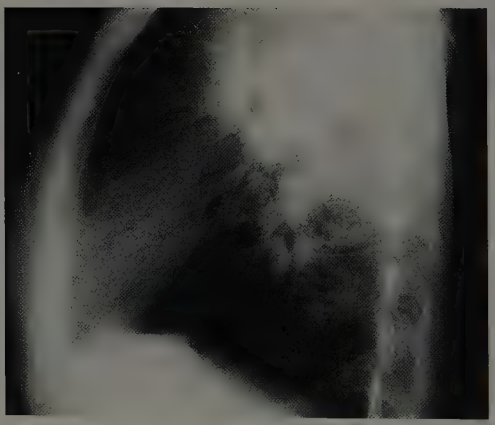

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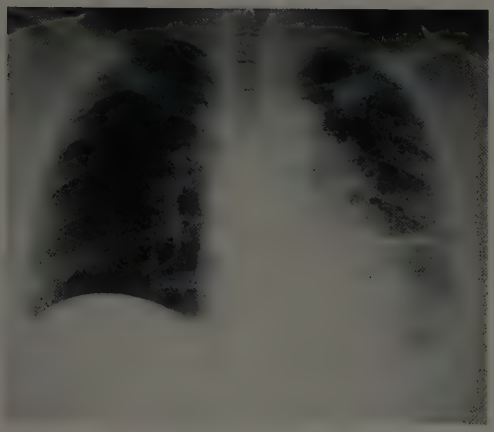
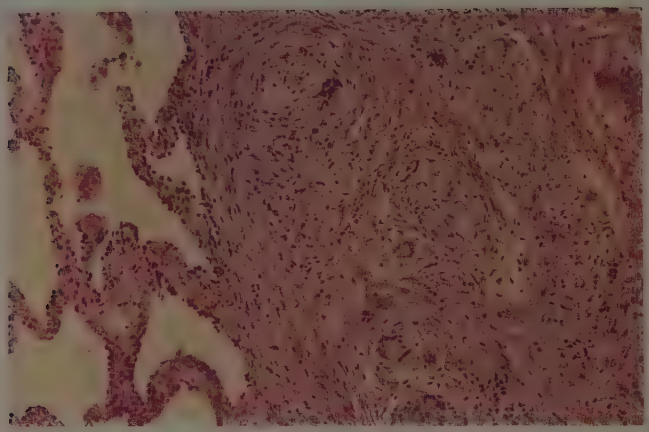


21

19 ♦ Extensive parenchymal disease due to sarcoidosis.

20 ♦ Adenopathy due to sarcoidosis. There is midzone upper lobe retraction and a coarse reticulonodular pattern.

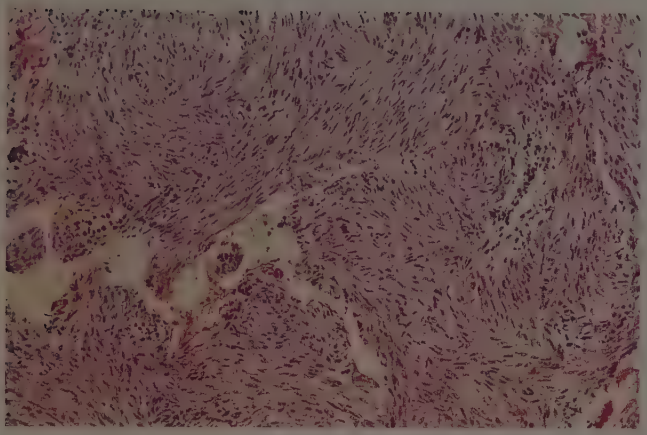
21 ♦ Unilateral sarcoidosis, a rare finding, is shown. Since sarcoidosis is usually symmetric and bilateral, an unusual unilateral presentation mandates a tissue diagnosis.



22

22 ♦ Pathologic specimens from areas involved with sarcoidosis typically demonstrate noncaseating granulomas. Characteristically, epithelioid and multinucleated giant cells make up the central core of the granuloma, interspersed with collagen.

23–24 ♦ Lymphangiomyomatosis. Microscopic evaluation of the lung parenchyma reveals accumulation of smooth-muscle bundles in the alveolar walls, especially around the bronchioles and venules.



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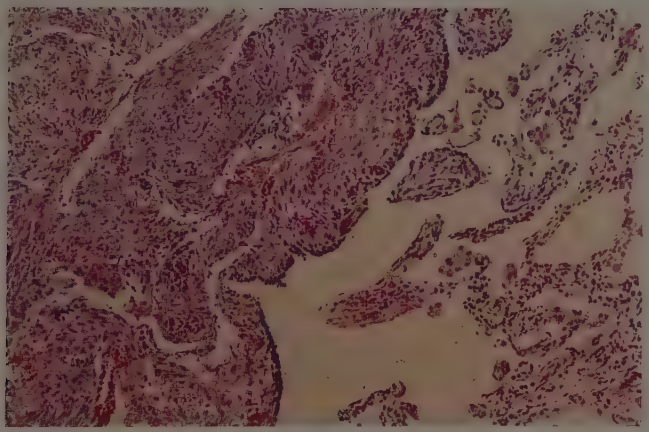

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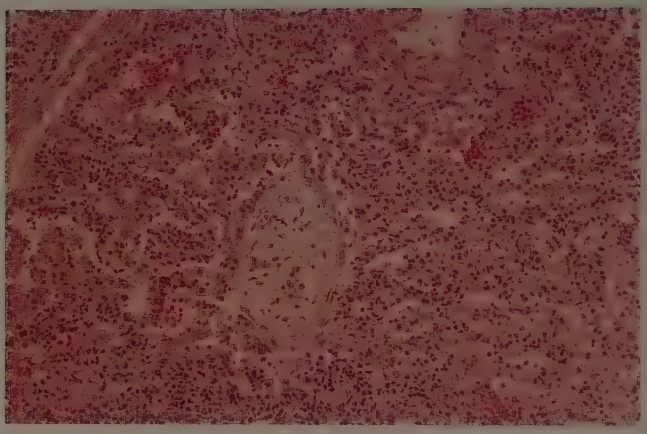

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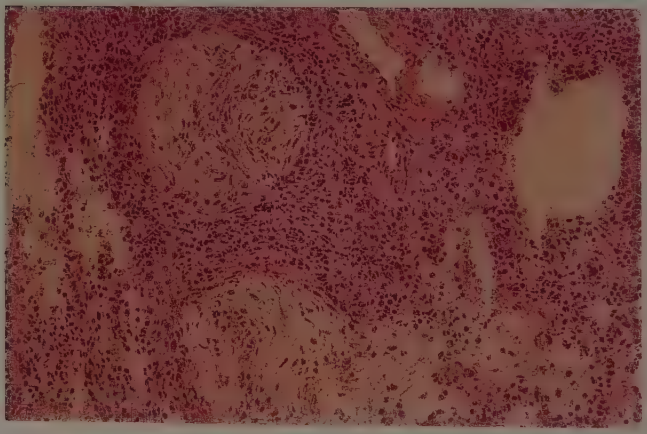

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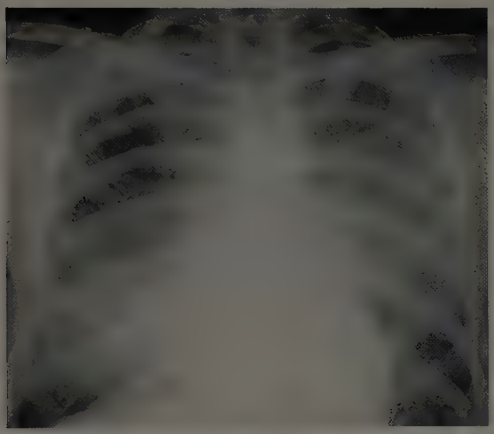
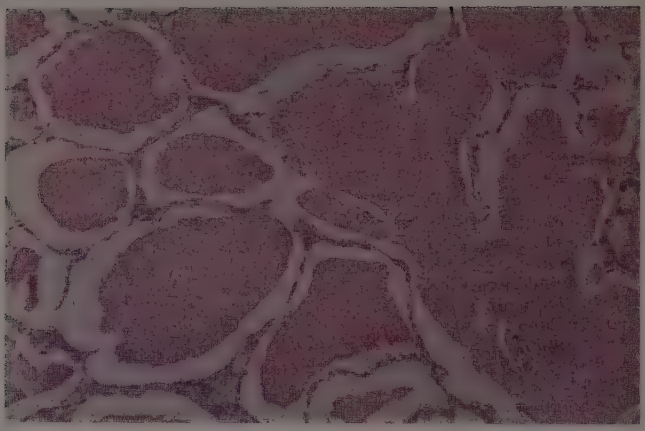


27

25 ♦ The most common finding of bronchiolitis obliterans and organizing pneumonia is patchy, nonsegmental airspace involvement with tufts of connective tissue in the bronchioles, alveolar ducts, and alveoli. This pattern is seen in patients with collagen vascular disease, drug reactions, organizing infections, hypersensitivity pneumonitis, and bone marrow transplant recipients.

26 ♦ Bronchiolitis fibrosa obliterans. Histologically there is plugging of the bronchiolar lumens and alveolar ducts by loose connective tissue or polypoid masses of granulation tissue.

27 ♦ Pulmonary alveolar proteinosis—a diffuse alveolar-filling process that can have the appearance of pulmonary edema.



28

28 ♦ The alveoli are filled with an insoluble, amorphus, granular, lipid material that stains positively with periodic acid-Schiff (PAS) stain. There is little inflammation. Findings are consistent with pulmonary alveolar proteinosis.

Clinical Pearls

1 ♦ There can be several different radiographic patterns seen in interstitial pulmonary fibrosis, including low lung volumes and increased interstitial markings predominantly in the lower zones, shadows throughout all lung fields, fine "ground-glass" appearance, and honeycombing in the most advanced cases.

2 ♦ The hallmark of eosinophilic granuloma is the proliferation of Langerhans cells known as histiocytosis X cells. Eosinophilic granuloma demonstrates histiocytic infiltration of lung, bone, or both.

3 ♦ Pneumothorax is common in eosinophilic granuloma and is often recurrent.

4 ♦ The lung volumes are generally normal or increased in the later stages of eosinophilic granuloma.

5 ♦ Chronic eosinophilic pneumonia (CEP) has some clinical and pathologic features which overlap other eosinophilic processes. However, the lack of an identifiable cause, radiologic findings, and exquisite responsiveness to corticosteroids are distinctive features of CEP. Corticosteroid therapy results in dramatic clinical and radiographic improvement within a few days of administration of treatment.

Continued on next page

6 ♦ Sarcoidosis is a multisystem granulomatous disease of unknown cause that frequently affects the lung. It is found predominantly in younger patients in the 20- to 40-year-old age group, and the usual presentation is bilateral hilar lymphadenopathy, pulmonary infiltrates, and skin or eye lesions. Sarcoidosis may involve the upper respiratory tract including the nasal mucosa, pharynx, and larynx. The intrathoracic involvement of sarcoidosis is usually staged as follows: stage 0, normal chest radiograph; stage 1, bilateral hilar lymphadenopathy; stage 2, bilateral hilar lymphadenopathy and parenchymal infiltrates; and stage 3, diffuse parenchymal infiltrates. An additional stage 4 is sometimes used to account for more advanced disease, including severe fibrosis and cavitation.

7 ♦ Lymphangiomyomatosis (an uncommon disorder seen only in women) is characterized by smooth-muscle proliferation throughout the lungs, leading to airway and interstitial disease, lymphatic obstruction leading to chylous effusions, and recurrent pneumothoraces. Lymphangiomyomatosis should be suspected in the setting of chylous effusions and recurrent pneumothoraces.

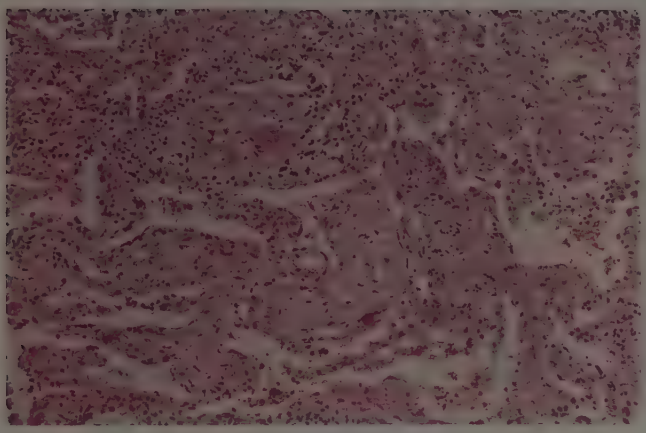
8 ♦ Bronchiolitis obliterans with organizing pneumonia is a distinct entity of infiltrative lung diseases. It is a common reaction pattern, especially in acute lung injury. The acute inflammation of small airways seen in bronchiolitis obliterans typically results in partial or complete obstruction of the bronchioles. The disease usually occurs after an acute and severe viral respiratory infection (commonly adenovirus) and is characterized by marked hyperinflation of the lungs.

9 ♦ Pulmonary alveolar proteinosis is a disorder of unknown etiology, found mostly in young and middle-aged patients, that presents as shortness of breath, hypoxemia, and a diffuse infiltrate on chest radiograph. Diagnosis is made by flexible bronchoscopy (FB) and bronchoalveolar lavage, which yields a characteristic milky fluid that is PAS positive, or by FB and biopsy.

29

29 ♦ Hypersensitivity pneumonitis. A diffuse acinar consolidation pattern with alveolar filling predominantly in the midlung zones; there is diffuse consolidation.

30–31 ♦ Histology shows infiltration of the alveolar walls with plasma cells, monocytes, histiocytes, and lymphocytes consistent with hypersensitivity pneumonitis.



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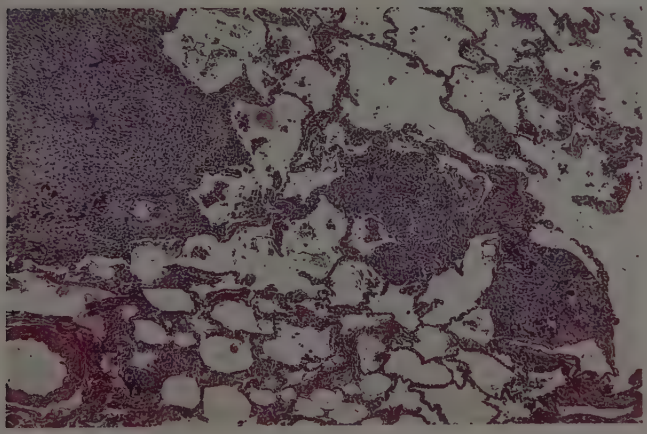

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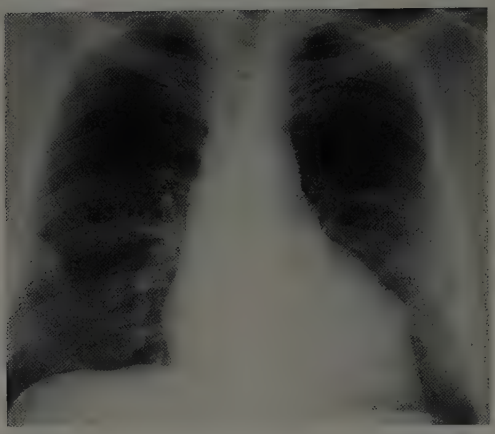
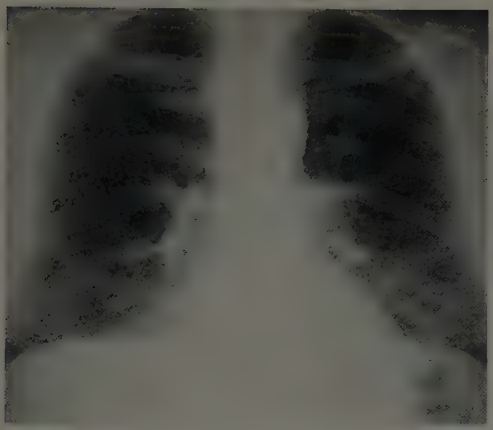


32

32 ♦ Asbestosis plaques on the left diaphragm and heart border.

33–34 ♦ Asbestosis can progress to severe interstitial fibrosis in the lower lung zones. This is manifested by a "shaggy" heart border and an ill-defined outline of the diaphragm with small irregular opacifications.



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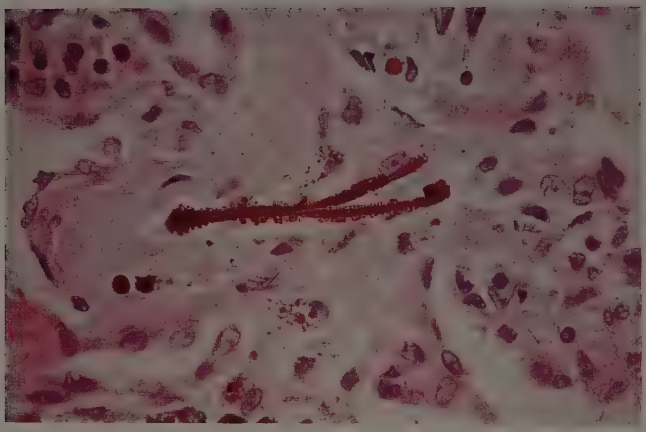

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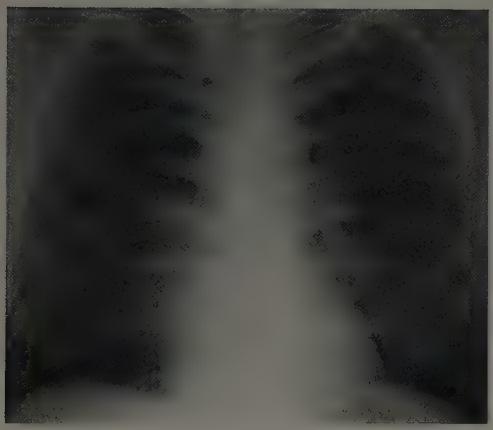
35 ♦ Asbestosis or ferruginous bodies may be seen microscopically in addition to fibrosis. These form within macrophages that have engulfed asbestos fibers.



36

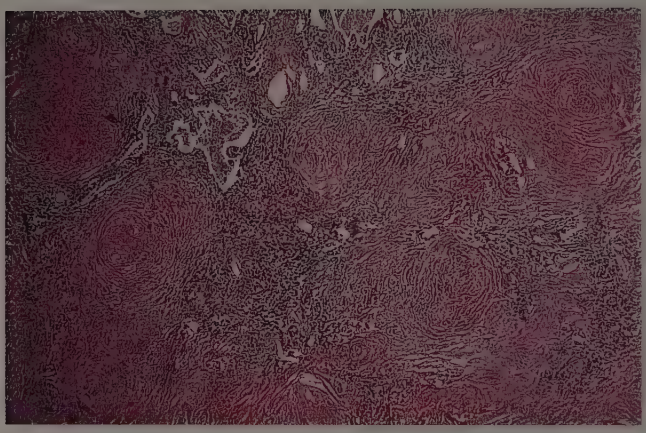
36 ♦ Berylliosis is seen as a fine reticulonodular pattern that spares the lung apices and bases.

37

37 ♦ Silicosis. Hilar eggshell adenopathy and honeycombing of the lung.

38

38 ♦ Hilar nodal calcifications due to silicosis are best seen on this oblique view. Silicosis may subsequently progress to massive fibrosis.



39

39 ♦ Silicotic nodules are characterized by a cell-free central area with concentric, whorled, hyalinized fibers around it, which are surrounded by connective tissue.

Clinical Pearls

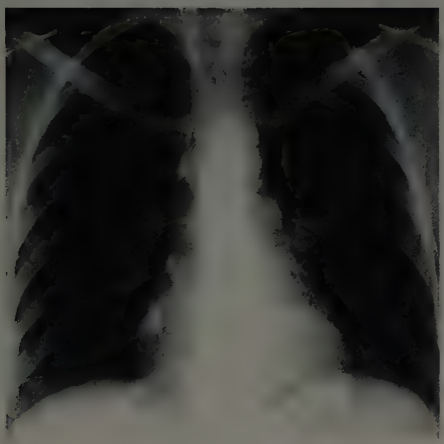
1 ♦ Hypersensitivity pneumonitis belongs to a group of granulomatous, interstitial, and alveolar-filling diseases resulting from repeated inhalation of and sensitization to a broad spectrum of organic dusts and low-molecular–weight chemical antigens. The illness is characterized by granulomatous pneumonitis that is reversible if the exposure is terminated. The prognosis for recovery is usually good with improvement of acute symptoms within a few days, and the disease is usually self-limited. However, a chronic form of hypersensitivity pneumonitis can develop.

2 ♦ The two major effects of asbestos exposure are fibrosis and tumor formation. In addition to the development of a diffuse pulmonary fibrosis (asbestosis), there is an increased risk of lung cancer and malignant mesothelioma of the pleura associated with asbestos exposure. The chest radiograph in asbestosis shows linear or irregular small opacities usually seen first in the lower lung zones with progressive loss of lung volume. The finding of diffuse or focal pleural thickening and/or focal hyaline pleural plaques along the lateral chest wall of the diaphragm should alert the physician to the possibility of asbestos-related disease. Asbestos exposure may present as benign, diffuse, calcified plaques on the pleural surface.

Continued on next page

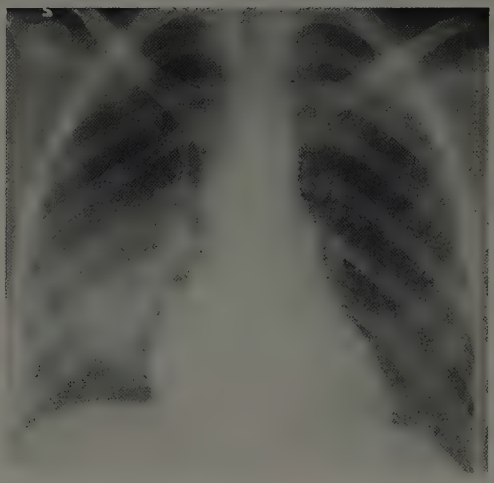
3 ♦ Beryllium enters the body by inhalation. The acute form of beryllium disease is uncommon and may range from irritation of the upper airways to pneumonia, bronchiolitis, or pulmonary edema. The chronic form of the disease usually occurs between 5 to 20 years after exposure and is associated with periodic remissions but ultimately progresses to pulmonary fibrosis. The disease usually presents as a granulomatous pneumonitis that progresses to pulmonary fibrosis. A late occurrence is honeycomb lung.

4 ♦ Silicosis refers to lung disease produced by respirable dust containing silicon dioxide (silica) in crystalline form. Three types of silicosis have been described: chronic, following decades of exposure; accelerated, following short, heavy exposure; and acute, following exposure to high concentrations. The chronic form produces disseminated, discrete, small hyalinized nodules throughout the lung. Silicosis with small foci of fibrosis is known as simple silicosis. The association between silicosis and tuberculosis has long been recognized. In some patients, the focal lesions coalesce to form masses of scar tissue known as progressive massive fibrosis.



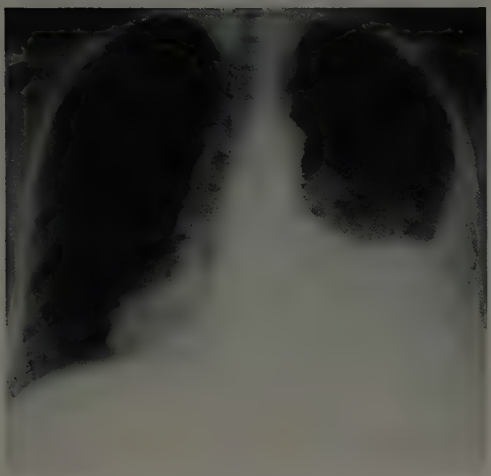
40

40 ♦ Prominent pulmonary artery segments secondary to pulmonary hypertension in a patient with systemic lupus erythematosus.



41

41 ♦ Systemic lupus erythematosus. Pericardial effusion, pleural effusion, and interstitial reticulations are shown.



42

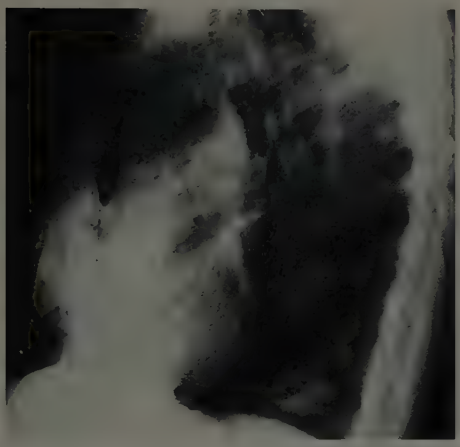
42 ♦ Pericardial and pleural effusion can be accompanied by lower lobe atelectasis in a patient with systemic lupus erythematosus. Impairment of ventilation due to diaphragmatic weakness results in the appearance of "shrinking lungs," manifested by elevated diaphragms and basilar atelectasis.

43

43 ♦ Lung disease in a patient with rheumatoid arthritis. There are diffuse reticulonodular opacifications with multiple well-circumscribed nodules of 3 to 4 mm.

44 ♦ The nodules, noted in 43, can be seen on the lateral film as well.

45 ♦ Multifocal ill-defined alveolar infiltrates with fine reticulations and multiple nodules in a patient with rheumatoid arthritis.



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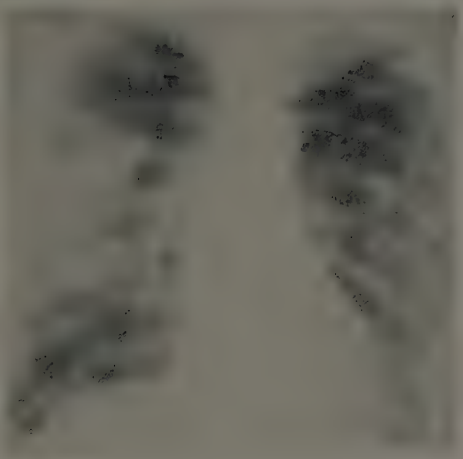
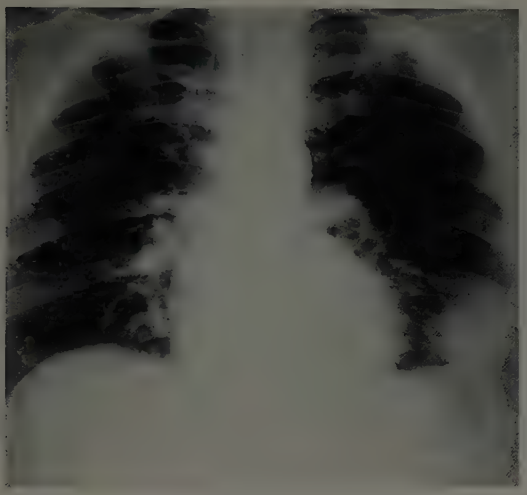


45

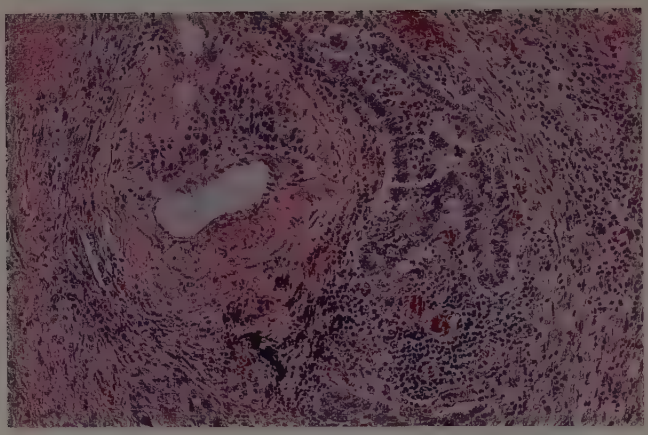
46

46 ♦ Wegener's granulomatosis can manifest radiographically as patchy alveolar infiltrates with thick-walled cavities.



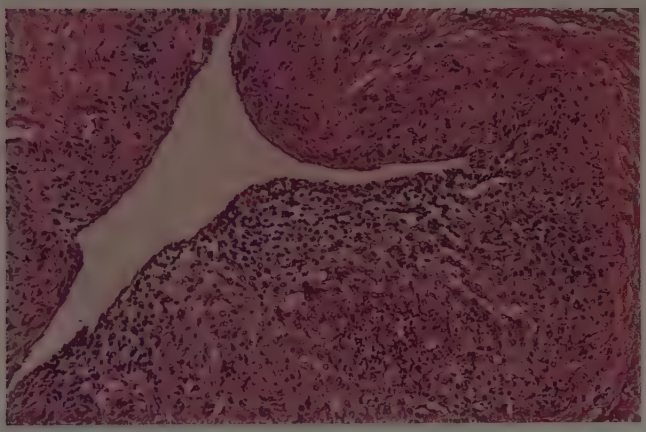
47

47 ♦ Single or multiple ill-defined, rounded infiltrates are also a radiographic finding of Wegener's granulomatosis.



48

48 ♦ Wegener's granulomatosis. Necrotizing vasculitis of small arteries and veins together with granuloma formation are the histopathologic hallmarks of this disease.



49

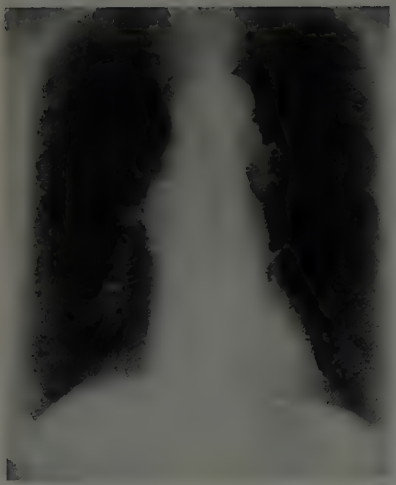
49 ♦ Wegener's granulomatosis.

Clinical Pearls

1 ♦ Diaphragmatic weakness in systemic lupus erythematosus is relatively common and can result in the appearance of "shrinking lung," manifested by elevated diaphragms and basilar atelectasis.

2 ♦ There are a number of pulmonary disorders associated with rheumatoid arthritis including: fibrosing alveolitis; necrobiotic nodules (with or without inorganic dust exposure); pleural effusions; obliterative bronchiolitis, and pulmonary vascular disease. Bronchiolitis obliterans increasingly has been reported in association with rheumatoid arthritis. The prognosis varies with the degree of lung involvement.

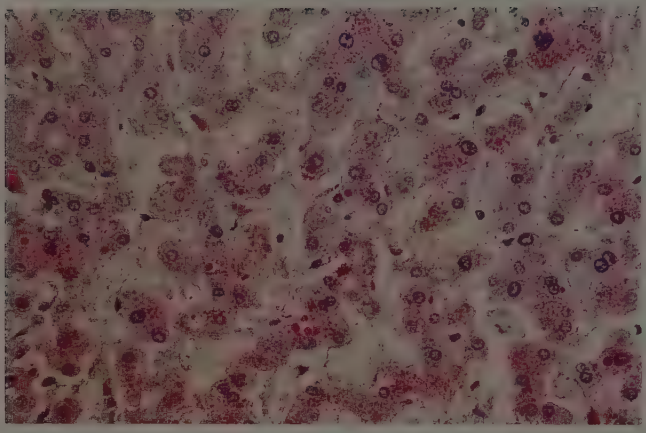
3 ♦ Wegener's granulomatosis is a necrotizing granulomatous vasculitis of both the upper and lower airways, glomerulonephritis, and small vessel vasculitis. The disease process can, however, be limited to the lung. Pleural effusions may be seen in 30% of patients.



50

51

50–51 ♦ α_1-Antitrypsin deficiency. Posterior-anterior and lateral views reveal clear lung fields, hyperinflation with flattened diaphragms and increased retrosternal air space, and a normal cardiac silhouette.

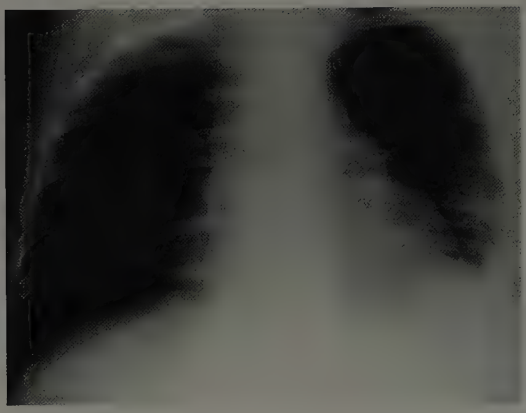


52

52 ♦ Lung biopsy demonstrates enlargement and coalescence of air spaces in a patient with α_1-antitripysin deficiency.

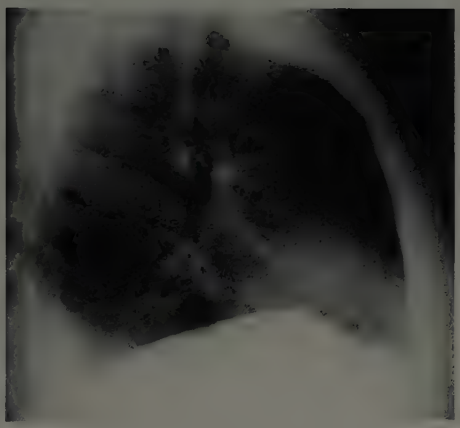
Clinical Pearl

1 ♦ Severe emphysema at a young age, lower lung zone emphysema, or emphysema in a nonsmoker should raise suspicion in the practitioner that specific testing for α_1-antitrypsin deficiency is indicated. α_1-Antitrypsin is an antiprotease that provides the major defense for the lower respiratory tract against the destruction by neutrophil elastase. α_1-Antitrypsin deficiency results in the loss of this protective mechanism, which may result in emphysema. The panacinar emphysema that is seen is most often more prevalent at the lung bases.



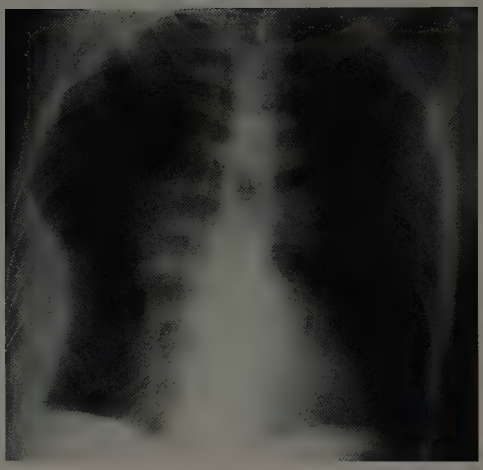
53

53 ♦ Left-sided pleural effusion that is subpulmonic and tracks up the lateral pleural space.



54

54 ♦ There is blunting of the costophrenic angle posteriorly, caused by pleural effusion.



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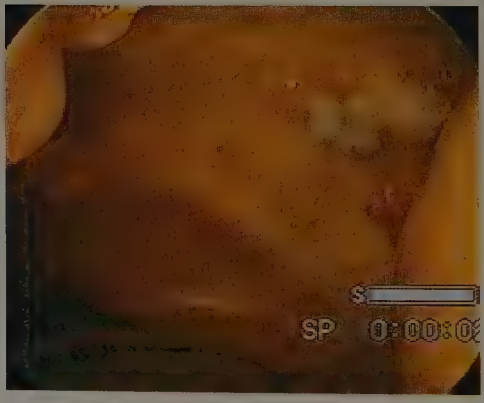

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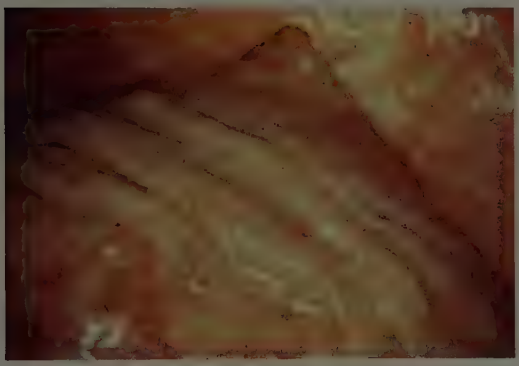


57

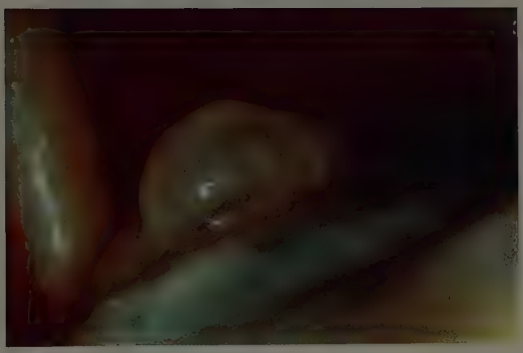
55 ♦ The pleural effusion may be confined to a defined space if loculated as seen on the right lateral aspect. A large effusion will generally shift the mediastinal structures contralaterally.

56 ♦ Thoracoscopic view of small-cell carcinoma. In order to diagnose the cause of pleural effusion, sampling of the pleura may be necessary.

57 ♦ Therapy is aimed at the inciting cause of the pleural effusion or may be done simply as a palliative measure (ie, pleurodesis). Pleurodesis should not be performed if the lung is "trapped" as shown and cannot expand.

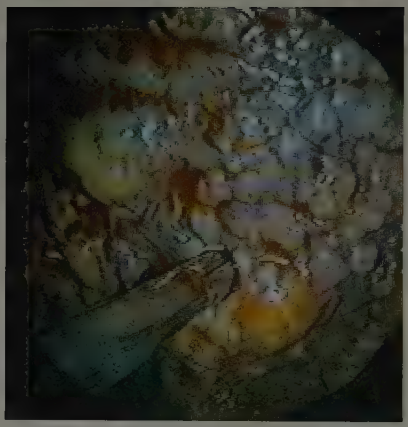
Clinical Pearl

1 ♦ Pleural effusions are classically divided into transudates and exudates by the criteria of Light applied to the yield of a thoracentesis. Transudates have a low protein or LDH relative to that of serum and exudates are high in these. The distinction is important in directing diagnosis and treatment. Transudates are often due to a systemic process, and treatment is aimed at the underlying disease. Exudates have a longer list of causes and may be due to a local pleural process or to a systemic disease. The patient may be asymptomatic or profoundly short of breath. Diagnosis typically begins with thoracentesis, which may also relieve dyspnea. If further tissue specimen is required, a blind pleural biopsy or thoracoscopy can be performed. In pleural tuberculosis, a blind biopsy with histology and culture should be diagnostic; whereas, pleural space malignancies may require thoracoscopy.



58

58 ♦ A recurrent pneumothorax or an unresolving first pneumothorax is managed by thoracoscopic control of blebs, possible pleurodesis, and, occasionally, thoracotomy.



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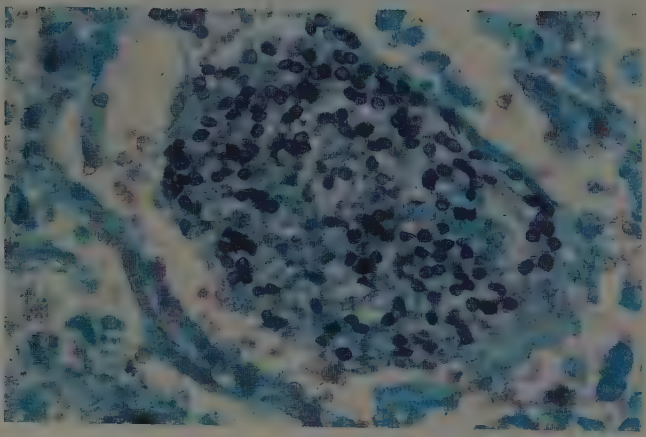


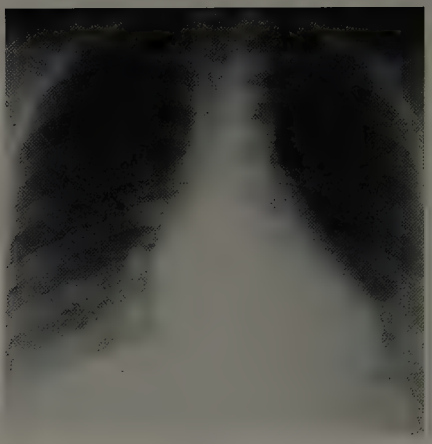
60

61

59 ♦ Thoracoscopic appearance of *Pneumocystis carinii* pneumonia-related pneumothorax.

60 ♦ Silver stain of the *Pneumocystis carinii* organisms. Secondary spontaneous pneumothorax is a more serious situation and even the initial episode requires tube thoracostomy. More invasive procedures are then dictated by the condition of the patient and the underlying disease.

61 ♦ Thoracoscopic view of talc distributed in the pleural space for pleurodesis with a chest tube in position immediately prior to lung reexpansion.



62

62 ♦ Typically, the chest radiograph will demonstrate a pleural line defining the edge between the collapsed lung and pleural space. A spontaneous right pneumothorax manifests radiographically as an absence of lung markings outside a defined lung edge laterally.

63 ♦ The pneumothorax may be irregular, and there may be a shift of the mediastinal structures to the contralateral side.

64 ♦ Left pneumothorax. The pneumothorax may be small and difficult to discern.

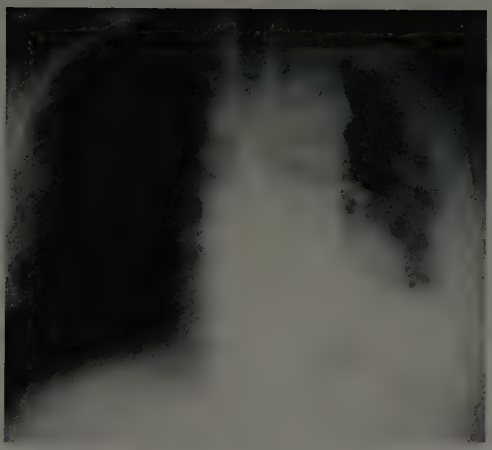
63

64

Clinical Pearl

1 ♦ Primary spontaneous pneumothorax occurs in patients without an apparent underlying disease. Chest pain on the affected side with dyspnea is the typical presenting complaint. This often occurs at rest and is rarely life threatening. Treatment depends on the extent of the pneumothorax, clinical symptoms, and if it is the initial episode.

Secondary spontaneous pneumothorax occurs in association with underlying lung disease. Although it most commonly occurs secondary to chronic obstructive pulmonary disease, many other conditions are associated. An initial pneumothorax of less than 15% in a hemodynamically stable patient may be managed with observation. If the pneumothorax is larger than 15%, it is managed by direct aspiration or tube thoracostomy.



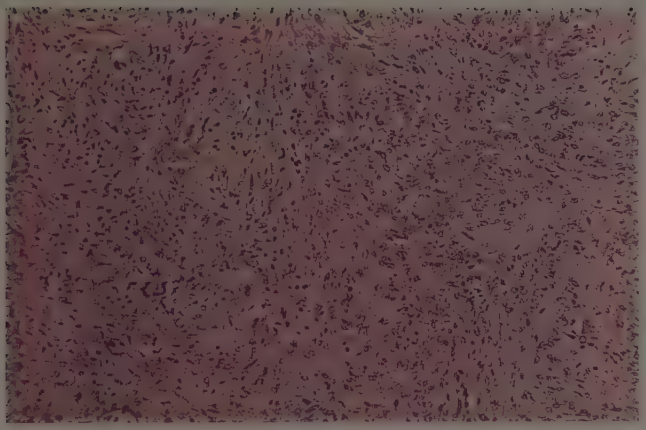
65

65 ♦ Malignant mesothelioma presents radiographically with a unilateral irregular thickening of the pleura that may be accompanied by ipsilateral effusion.

66

66 ♦ The pleural effusion associated with malignant mesothelioma may be small or progress to total opacification of the hemithorax. Mediastinal shift to the contralateral side is limited in this case by fixation of the structures with a pleural ring of neoplastic tissue.

67-68 ♦ Microscopically, the malignant mesothelioma is classified as epithelial, mesenchymal, or mixed mesothelioma.



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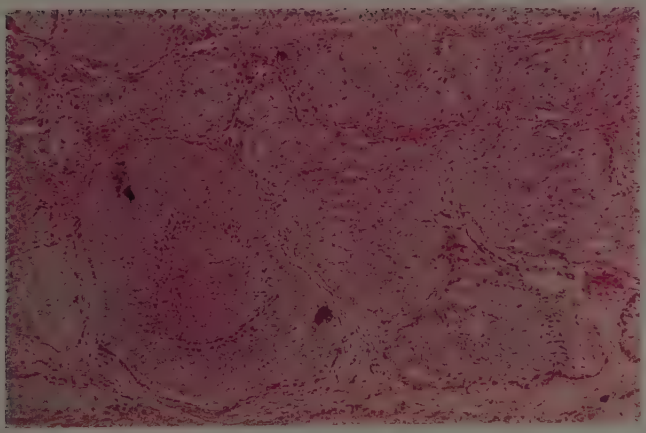
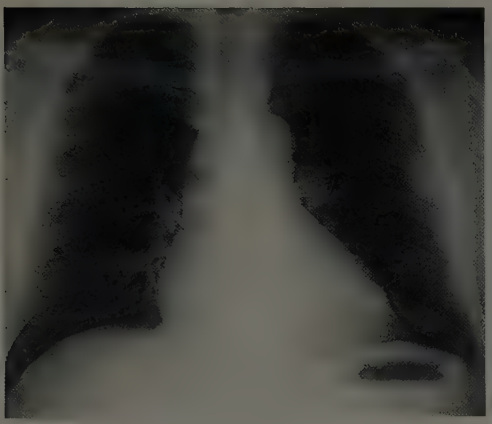


68

69

69 ♦ Thoracoscopic view of pleural plaques found in malignant mesothelioma.

70-71 ♦ Benign fibrous mesothelioma (benign fibrous tumor of the pleura). Isolated pleural nodules that were diagnosed at surgery as benign fibrous mesothelia.



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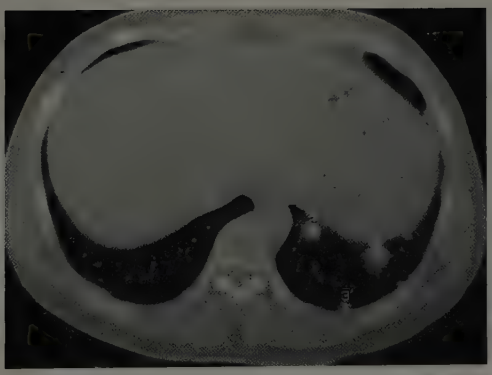

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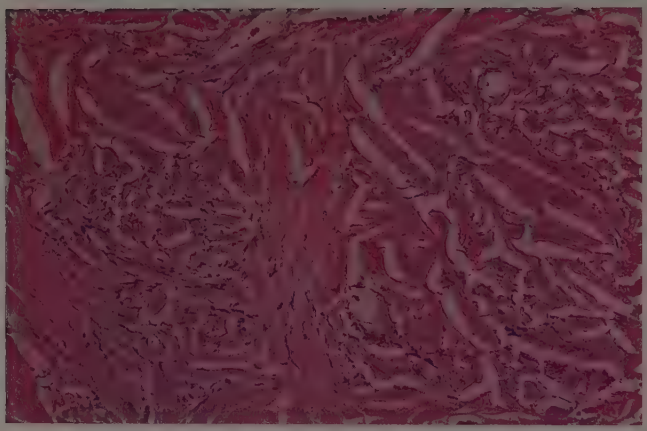


72

72 ♦ Benign fibrous mesothelioma is usually seen as a broad, white, fibrous pleural plaque. Microscopically, there is dense collagen without inflammation. No mitoses or atypia are seen.

Clinical Pearls

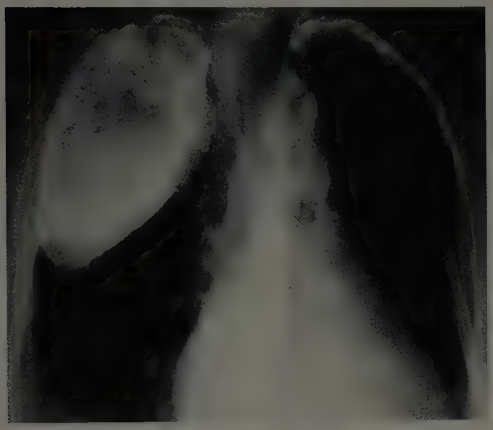
1 ♦ As a chest tumor, malignant mesothelioma is a tumor of the pleural cavity that can initially appear as discrete nodules and progress to total encasement of the pleural cavity. The tumor arises from the mesothelial lining cells and the patient has an extremely poor prognosis. Patients present in their fourth to seventh decades and at least 50% of patients have a history of asbestos exposure, with the time from exposure until diagnosis being 20 to 40 years. The most common clinical manifestations are chest pain, dyspnea, and a large exudative pleural effusion. Diagnosis is sometimes made with a blind pleural biopsy but often requires thoracoscopy or thoracotomy. Differentiation from adenocarcinoma may require special staining. Electron microscopy may supplement standard light microscopy. Prognosis is poor and treatment is for palliation in most cases, although experimental protocols are available.

2 ♦ Benign fibrous mesothelioma is a rare condition that arises from both visceral and parietal pleura and is not related to asbestos exposure.

73

73 ♦ Pleural tuberculosis. Unexplained exudative effusions often predominate with lymphocytes, and the diagnosis is most commonly made by pleural biopsy that shows caseating granulomata and yields the organism in culture.

74-75 ♦ The chest radiographic findings are similar to those of other unilateral pleural effusions. Posterior-anterior and lateral views demonstrate a calcified oleothorax. The patient had been treated for tuberculosis (TB) prior to the availability of anti-TB chemotherapy with a therapeutic collapse of her right lung using oil that was left in place and subsequently had calcified.



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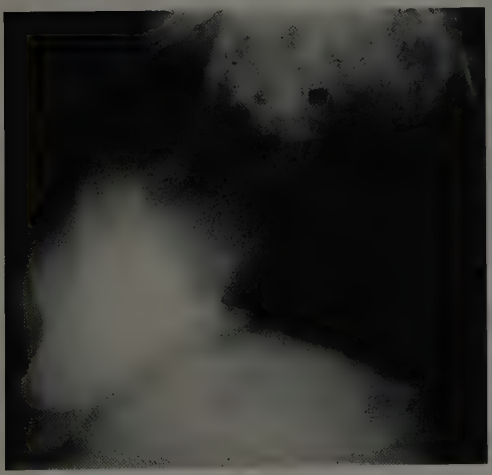
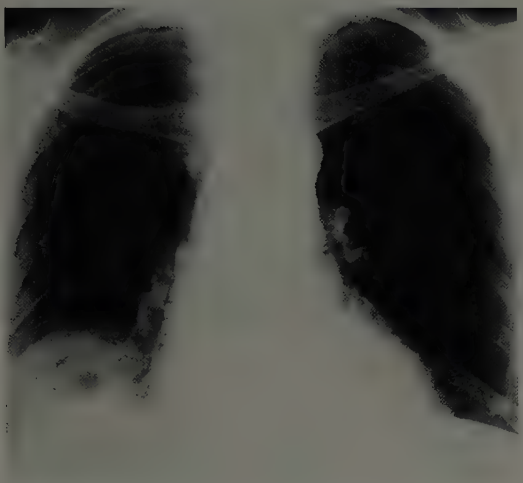


75

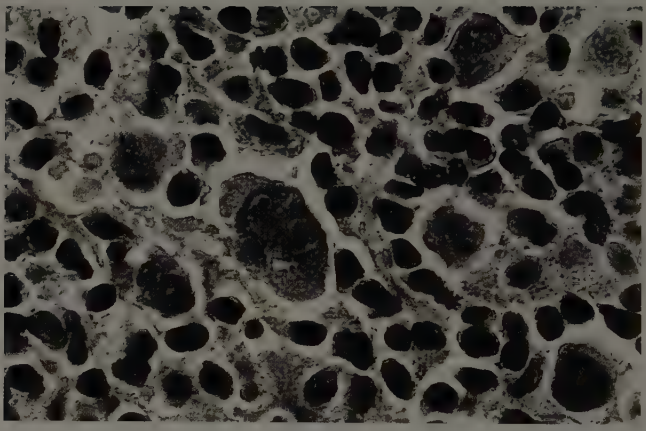
Clinical Pearl

1 ♦ Also known as tuberculous pleurisy, pleural tuberculosis is probably an inflammatory response to tuberculous protein released into the pleural space from a ruptured parenchymal site of infection. It may not be associated with simultaneous parenchymal disease and can be self-limited. It must be suspected in cases of unexplained exudative effusions. Although the pleurisy is often self-limited, the patient requires treatment as the risk of developing active tuberculosis is high.



76

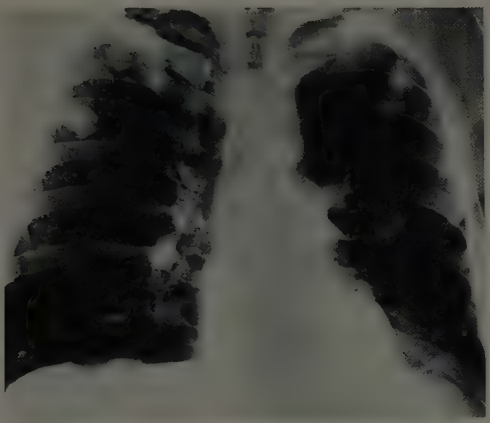
76 ♦ Non-Hodgkin's lymphoma. A 4×5-cm cavitary mass with shaggy borders in the right lower lobe. There is no associated hilar adenopathy.



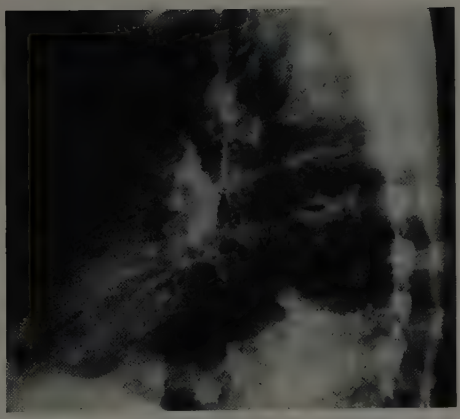
77

77 ♦ The bronchoscopy performed on the patient in 76 revealed a large submucosal mass completely obstructing the right bronchus intermedius. The overlying mucosa was granular. Pathologic studies of the biopsy material reveal chronic inflammation.

78-79 ♦ Radiographically, lymph node infiltration, reflected by hilar or mediastinal node enlargement or both, is the most frequent intrathoracic manifestation of non-Hodgkin's lymphoma. Pleural, bronchial, and pulmonary parenchymal disease is also seen.



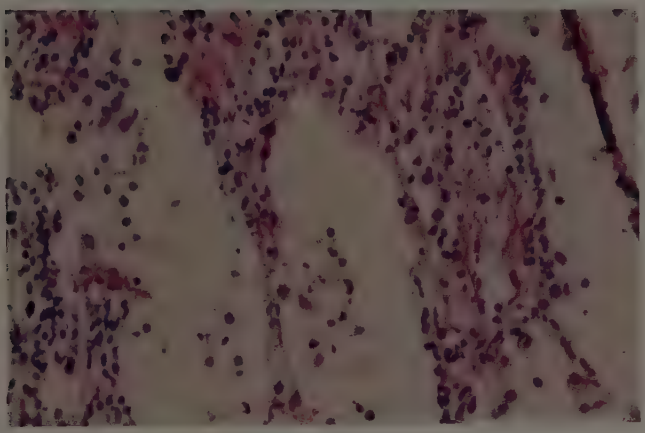
78



79

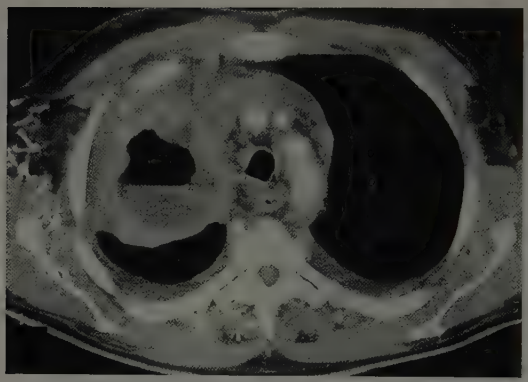
80

80 ♦ A pattern of small irregular opacities throughout both lungs in a patient with non-Hodgkin's lymphoma. Flat diaphragms and large lung fields consistent with chronic obstructive lung disease are also evident.



81

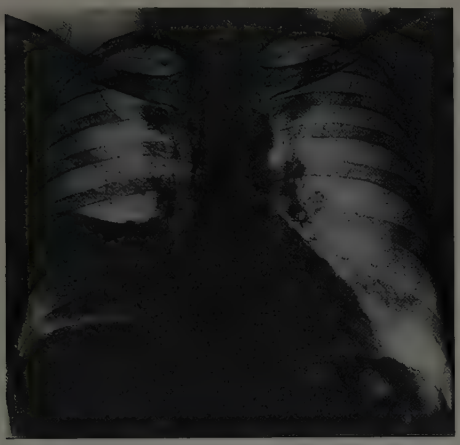
81 ♦ The lung biopsy shows the interstitial tissue to be focally thickened by collections of small round lymphocytes. The cells are relatively uniform with slightly irregular nuclei. These cells are infiltrating the submucosa and the lymphatics, and stain positively with immunohistochemical stains for leukocyte-common antigen and B-lymphocyte cell markers. These biopsy findings are consistent with malignant lymphoma of mucosa-associated lymphoid tissue (MALToma).



82

82 ♦ Squamous cell carcinoma. Computed tomography scan with contrast of the chest shows a large necrotic right upper lobe mass extending to the mediastinum encasing the trachea and right and left bronchi. The right upper lobe bronchus is not visible, and the mass obstructs the innominate vein; the superior vena cava and multiple vascular collaterals are also seen.

83-84 ♦ Radiographic presentation of squamous cell carcinoma in a different patient reveals a right middle lobe density; however, there are no visible air bronchograms, and the medial part of the horizontal fissure, in the posterior-anterior view, curves downward. These findings suggest volume loss of the medial segment of the middle lobe due to obstruction and a postobstructive pneumonia.



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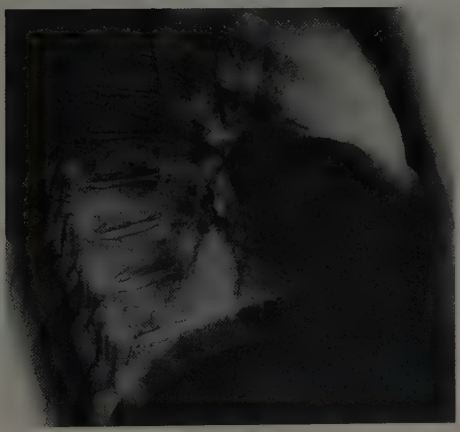


84

85

85 ♦ A cavitary lesion is seen as part of the radiographic presentation of squamous cell carcinoma. In addition, there are infiltrates in the right upper lobe, a prominent fissure, and markedly enlarged right paratracheal and hilar lymph nodes.

Clinical Pearls

1 ♦ While pulmonary involvement in Hodgkin's disease is common (involving up to 40% of patients), primary pulmonary Hodgkin's disease presenting as an isolated lung mass is relatively uncommon.

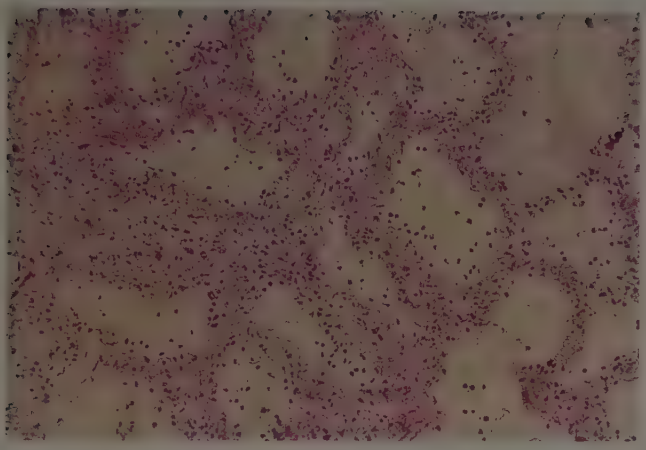
2 ♦ Since noninvasive studies are rarely diagnostic, open lung biopsy is usually required to confirm the diagnosis of Hodgkin's disease. Needle aspiration biopsy often fails to provide adequate material for diagnosis.

3 ♦ Lymphoma arising in the bronchial (mucosal)-associated tissue (BALT/MALToma) can be limited to the lung with no extrapulmonary adenopathy. MALToma can present as chronic diffuse interstitial lung disease, similar in most features to other chronic diffuse interstitial lung diseases.

4 ♦ Squamous cell carcinoma of the lung most commonly presents as a central lesion involving a major or lobar bronchus. As it increases in size, the tumor causes obstruction and atelectasis. Squamous cell carcinoma of the lung may cavitate.

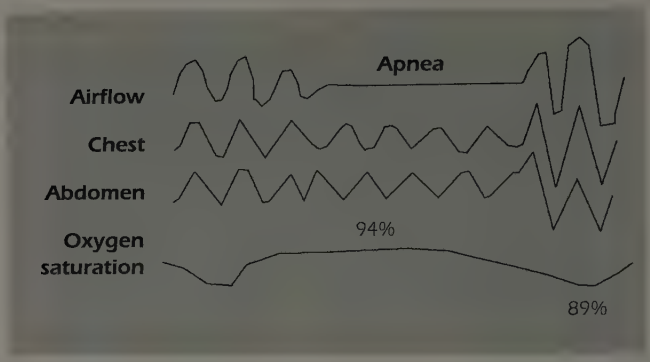
86

86 ♦ The radiographic appearance of adult respiratory distress syndrome is that of a diffuse alveolar-filling process. The radiographic findings must be correlated with the clinical picture to make the diagnosis.



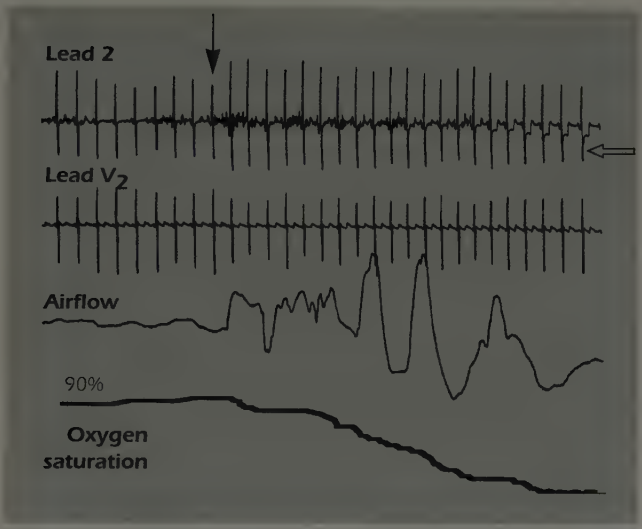
87

87 ♦ Adult respiratory distress syndrome. Diffuse alveolar damage with hyaline membranes, interstitial edema, and inflammation. This may progress to fibrous organization.



88

88 ♦ Sleep disorder. On polysomnography, airflow is monitored to detect apnea (absence of airflow >10 s) or hypopnea (reduction in airflow). Chest and abdominal movements are also monitored. In obstructive apnea, the chest and abdomen move during apnea (persistent inspiratory effort). The arterial oxygen saturation is monitored by oximetry. The anterior tibialis electromyography of both legs is recorded to detect periodic leg movements.



89

89 ♦ Polysomnography shows severe obstructive sleep apnea syndrome demonstrated by multiple episodes of obstructive apneas, lasting up to 1 minute in length, associated with severe oxygen desaturation and multiple arousals. The apnea index (number of apneas per hour of sleep) is 85. The percent sleep time with an oxygen desaturation of <90% is 67%. In addition, the occurrence of ST-segment depression in association with minimum oxygen saturation following apnea termination is observed. In the example, apnea termination occurs at the vertical solid arrow. Following apnea termination, there is an increase in heart rate, continued desaturation, and the appearance of ST-segment depression in lead II (horizontal open arrow), which abates by the onset of the next apnea.

Clinical Pearls

1 ♦ Adult respiratory distress syndrome is a clinical syndrome characterized by noncardiogenic pulmonary edema and severe hypoxemia. There is a systemic inflammatory response that leads to an increased pulmonary capillary permeability.

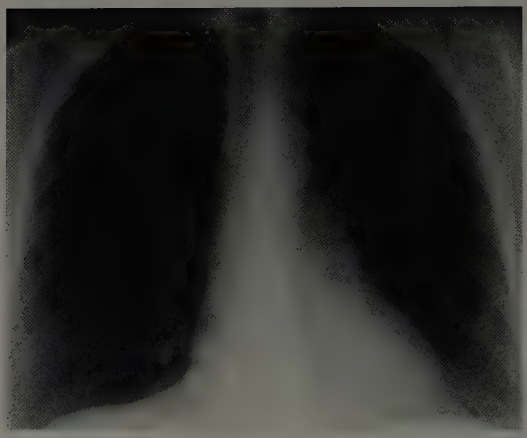
2 ♦ The diagnostic procedure of choice for excessive daytime sleepiness is a sleep study (polysomnography). During this procedure, selected electroencephalographic leads, eye movements, and chin electromyograms are monitored to detect the presence and the stage of sleep. The standard variables in a sleep study are monitored to evaluate excessive daytime sleepiness. If there are also complaints of nocturnal chest pain, three electrocardiogram leads (I, II, V_2) are also placed.

3 ♦ Nocturnal ischemia, frequently "silent" clinically, may have its origin in sleep-disordered respiration. Patients with nocturnal ischemia should be questioned about symptoms of sleep-disordered breathing, especially snoring and excessive sleepiness.

90

91

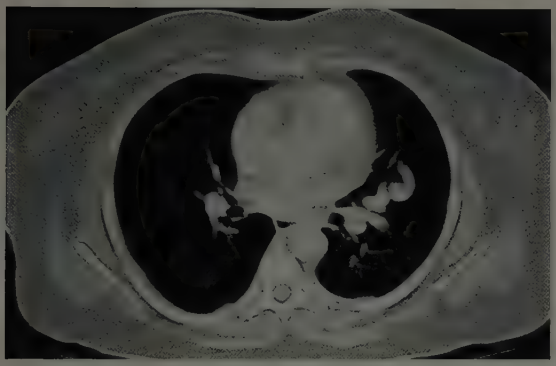
90-91 ♦ Yellow nail syndrome is a triad of slow-growing discolored nails, lymphedema involving head or extremities, and exudative pleural effusion that is also often associated with bronchiectasis.



92

92 ♦ Congenital arteriovenous malformation. Typical radiologic findings include a well-defined lower lobe mass with feeding vessels—artery and vein.

93-94 ♦ Computed tomography scans of patient in 92.



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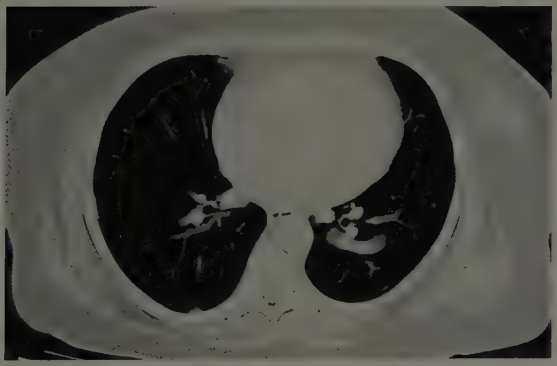

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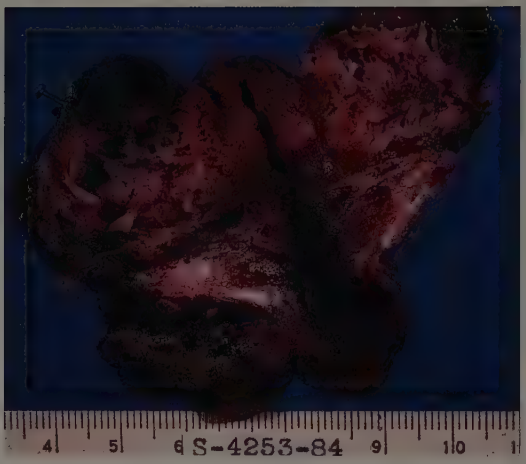
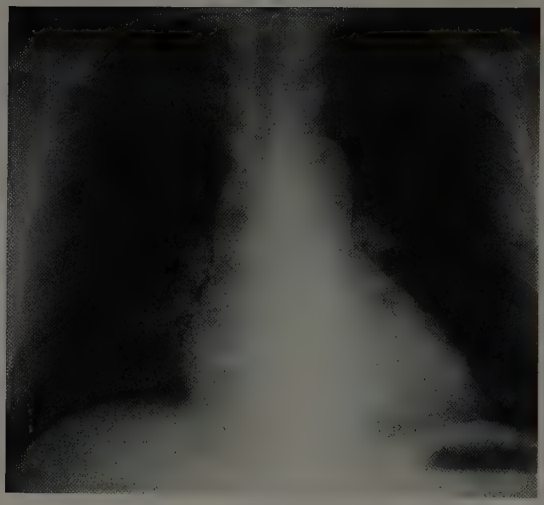


95

95 ♦ Congenital arteriovenous malformation found in the rare case of resection.

96

96 ♦ Congenital arteriovenous malformation at autopsy.

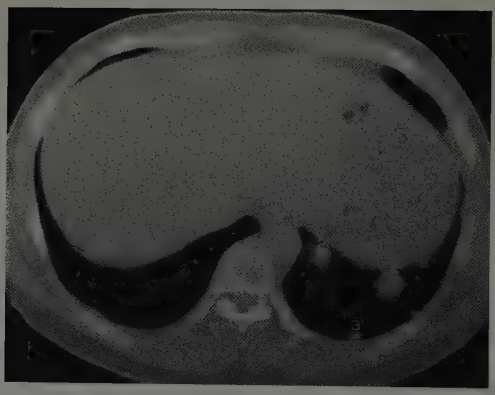


97

97 ♦ Patient is a 54-year-old man who had sustained a gun shot wound to the abdomen as a young man, with resultant thoracic splenosis. The posterior-anterior view demonstrates a large nodule in the left chest.

98

98 ♦ Thoracic splenosis. Lateral view of patient in 97 showing large nodule.



99

99 ♦ The chest computed tomography of the patient in 97 and 98 demonstrates multiple nodules consistent with thoracic splenosis.

Clinical Pearls

1 ♦ Yellow nail syndrome (approximately 100 reported cases) is a rare triad of slow-growing nails, lymphedema involving the head or extremities, and exudative pleural effusion that is often associated with bronchiectasis. Yellow nail syndrome is not related to cancer or connective tissue disease. Lymphangiograms have shown lymphatic hypoplasia. The pleural effusions are usually bilateral and rarely remit spontaneously. Pleural biopsy may show fibrosis or lymphocyte infiltration but is nonspecific. The effusion is caused by impaired lymphatic drainage from the pleural space, and fluid reaccumulates rapidly after thoracentesis. It should be noted that yellow nails are not always present.

2 ♦ Congenital arteriovenous malformations and some hereditary disorders are characterized by one or more abnormal communications between pulmonary arteries and veins causing a right-to-left shunt. Oxygenation is compromised since blood flowing through this abnormal communication does not get oxygenated. In 50% of cases, the problem is limited to the pulmonary system; in the remaining 50% of cases, it can be associated with a systemic hereditary disorder known as Osler-Weber-Rendu disease or hereditary hemorrhagic telangiectasia.

Continued on next page

3 ♦ Thoracic splenosis is an entity that results from trauma-induced implantation of splenic tissue into the thoracic cavity. The splenic tissue implants onto the pleural surface, derives its own blood supply, and functions as normal splenic tissue. Diaphragmatic rupture is not always found. The lesion is usually asymptomatic, although pain is reported in some patients. Typically a number of years will elapse between the time the trauma is sustained and the lesion is diagnosed; usually by chest radiograph. The chest radiograph or computed tomography may show multiple solitary nodules or a mediastinal mass. The lesions may range in size from a few millimeters to 7.5 cm.

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